MERIT RESEARCH JOURNALS
www.meritresearchjournals.org

Merit Research Journal of Medicine and Medical Sciences (ISSN: 2354-323X) Vol. 6(6) pp. 215-218, June, 2018
Available online http://www.meritresearchjournals.org/mms/index.htm

Original Research Article

Does obesity cause microalbuminuria?

Osama Mirghani Osman[1*], Meshal Abdulrahman Hassan Ogran[2], Faisal Shaleh Alotaibi[2] and Haider Ali Omer Mohamed[3]

[1]Associate Professor, Department of Physiology, College of Medicine, King Khalid University, Abha, Saudi Arabia.

[2]Medical Student, College of Medicine, King Khalid University, Abha, Saudi Arabia.

[3]Associate Professor, Consultant Nephrologist, College of Medicine, King Khalid University, Abha, Saudi Arabia.

***Corresponding Author's**
Email:Osama_elneel@yahoo.com
Mobile: 00966536062554

Abstract

The objective of this study was to find whether obesity and its various indices has a detrimental effect on the kidney function as manifested by microalbuminuria. 120 young male adults were included. They were divided into two groups, normal with BMI between 18 and 25 and the obese group with BMI more than 30. Exclusion criteria include subjects with hypertension or diabetes. For each subject weight, height, waist circumference, hipline and fat % were measured. Urine sample was collected from each subject for measurement of microalbumin and creatinine. No significant difference in the incidence of microalbuminuria between the normal and the obese subjects was found. No correlation was found between the level of microalbuminuria and the various indices of obesity. We conclude, obesity does not cause microalbuminuria in young adult obese males who are otherwise healthy.

Keywords: Obesity, Microalbuminuria, Young adults, Non-diabetic, Normotensive.

INTRODUCTION

Obesity is one of the biggest health hazards. It is becoming a worldwide epidemic. According to WHO report obesity contributes to 2.6 million deaths worldwide every year (WHO report, 2005). Too much intake of energy, genetic susceptibility, as well as physical inactivity are the major causal obesity factors, whereas endocrine disorders, gene mutations, psychiatric illnesses or medication might be the underlying causes in certain instances (Kaila and Raman, 2008). In the developing world the incidence of obesity is increasing with the changing of life style affecting these societies (Bhurosy and Jeewon, 2014)

A number of the population-based researches has demonstrated that there is a link between the measures of obesity as well as the progression and development of chronic kidney disease (CKD) (Kovesdy *et al*., 2017). High BMI generally appears to be linked to the presence, as well as the development of lower estimated rate of glomerular filtration, with a rapid loss of the estimated glomerular filtration rate(GFR) and development of end stage renal disease (ESRD) as well as the progression of CKD in patients having pre-existing CKD (Rifkin *et al*.,2010).

Obesity is a documented significant contributor of increased diabetes mellitus, cardiovascular disease and hypertension. (Poirier *et al*., 2006). All of these risks are capable of promoting chronic kidney disease. There has been an increasing appreciation that even without these risks, obesity itself largely increases CKD besides accelerating its progression (Coresh *et al.,* 2007). Previous researches indicated that high body mass index forms one of the greatest risk factors for new-onset chronic kidney disease (Jafar *et al*., 2009). There are different postulated mechanisms by which obesity may induce renal malfunction. In people who are affected by obesity, there is the occurrence of compensatory hyperfiltration in order to meet the heightened metabolic demands of the rise in the body weight. The rise in

Table 1. Shows the distribution of age, weight, height, BMI, waist circumference, hip line, waist to hip ratio % of fat and MCR for both normal and obese groups

	Normal(n=59)	Obese(n=61)	P value
Age(years)	21.8±1.6	21.9±1.8	NS
Weight(kgs)	64±9.1	111±14.1	<0.001
Height(cm)	170.4±7.8	171.6±5.5	NS
BMI	21.8±2.3	37.7±5.3	<0.001
Waist circumference (cm)	70.6±17.6	103.8±24.9	<0.001
Hip line(cm)	78±17.9	107.9 ±25.7	<0.001
Waist to hip ratio	0.9±0.1	0.96 ±0.0	<0.001
Fat %	13.2±5.5	33.4 ±4.7	<0.001
MCR	12.2	12.1	NS

The significant level was taken with P value less than 0.05.NS: not significant

intraglomerular pressure is capable of damaging the kidneys besides increasing the risk of the development of CKD within the long term (Brantsma *et al.,* 2007). Other adverse effect of obesity on renal function includes increased levels of acute phase reactants, and cytokines as well as reactive oxygen species (Sonta *et al.,* 2004). The hormone leptin and other fat derived hormones which are found to be elevated in obese subjects was found to accelerate the scarring within the kidneys (Bullo *et al.,* 2003). Histologically, focal segmental glomeruosclerosis is the main documented obesity related kidney damage (Kambham *et al.,* 2001). There are evidences showing that targeting obesity should benefit in maintaining and preserving kidney function (Agnani *et al.,* 2005).

This study was therefore undertaken to:

a. Determine whether microalbuminuria is a manifestation of obesity.

b. Determine any possible relationship of the indices of obesity namely body mass index (BMI), waist circumference, hipline, waist to hip ratio and fat % to microalbuminuria as indicated by microalbumin creatinine ratio(MCR).

SUBJECT AND METHODS

This study was approved by the ethical committee of the college of medicine at king Khalid University, Abha, Saudi Arabia. All participants provided written informed consent. The study was conducted over 3 months in the period from Feb. 2017 to April, 2017. Subjects were randomly selected from university students who agreed to participate. Only those with BMI between 18 and 24.9 and those with BMI over 30 were chosen. A total of 120 subjects were included. Subjects with history of diabetes or hypertension were excluded. For each subject the following measurements were performed; weight, height, waist circumference, hipline and % of fat. anthropometric measurements recorded with non-elastic tape using standard methods. The body mass index (BMI) was calculated (as kg/m^2). Waist to hip ratio was calculated. A fresh urine sample was collected from each subject for measurement of microalbumin (mg/L) and creatinine (g/L) and used for calculation of microalbumin creatinine ratio (MCR). A semi quantitative method (Microalbumin 2-1 ComboStrip) was used for measurement of urinary microalbumin and creatinine. The kit was provided by CLIA*waived*™

Statistical analysis

SPSS version 20 (IBM Corp., Armonk, NY) was used for standard statistical analysis. Descriptive data were presented as mean ± standard deviation. The Student's t-test was used to compare the two means. Spearman's correlation coefficient was used to test for association between MCR and indices of obesity. The significant level was taken with P value less than 0.05.

RESULTS

There was no significant difference in the age and height between the normal and obese group. All indices of obesity were significantly higher in the obese group than the normal group

No significant correlation was found between the level of microalbuminuria as indicated by MCR and the BMI, waist circumference, hipline, waist to hip ratio and fat % in both the normal and obese group.

Table 2. Shows the correlation analysis between MCR and the various indices of obesity in both the normal and obese group

Parameter	Normal(n59)		Obese(n60)	
	r value	P value	r value	P value
BMI	-.05	.70	.14	.27
Waist circumference	-.14	.28	-.17	.19
Hipline	-.13	.35	-.19	.15
Waist/hip ratio	-.05	.71	.12	.35
%fat	.02	.89	.18	.17

The significant level was taken with P value less than 0.05.

DISCUSSION

Obesity may be the most preventable risk factor for CKD. This may be due to the strong link of obesity with diabetes and hypertension, the two primary causes of CKD (USRDS 2005) as well as the postulated direct effect of obesity on renal function.

Screening for microalbumin in urine may be the most effective way to early identify subjects who are at increased risk for renal abnormalities (Koroshi, 2007). Consequently, early detection and correction of microalbuminuria is a key for preventing deterioration of renal function.

In this study we aimed at finding whether obesity as such may affect the renal function as manifested by urinary microalbumin excretion and if there was any abnormality in kidney function was it related to a particular parameter of obesity in otherwise healthy individuals.

Our results showed no significant difference between the urine findings from obese subjects and the normal subjects regarding MCR (Table I). Also there was no correlation between microalbuminuria and various indices of obesity in both normal and obese group including BMI, waist circumference, hipline, and fat % (Table 2).

Yesim *et al.* (2007) reached the same conclusion from their study on obese non diabetic non hypertensive females. The age group of their subjects was around 34 years. Similarly, Hasit and Vasudha (2012) found no microalbuminuria in obese non hypertensive non diabetic subjects with a wider age group range. Hoffmann *et al*. found that microalbuminuria is not related to BMI or waist to hip ratio in a group of glucose tolerant Hispanics (Hoffmann *et al.,* 2001). The above studies and our findings are in contrast with other studies that linked obesity to microalbuminuria. Chen *et al*. (2010) reported an increasing incidence of microalbuminuria in obese subjects but mainly in patients who are above 60 years and especially if the subjects have feature of metabolic syndrome. Similarly, a study on Chinese subjects (Lin *et al*.,2012) found an independent relationship between waist/hip ratio and the prevalence of microalbuminuria, which we could not detect in this study as shown by the non-significant correlation between MCR and both waist circumference and waist/hip ratio (table II). As in the study by Chen *et al.* (2010) the average age of their subjects was 60 years. Aging is a known cause of endothelial dysfunction which is one of the mechanisms that leads to microalbuminuria (Douglas *et al.,* 2011). A high prevalence of microalbuminuria was also detected in Subjects with insulin resistance syndrome independent of hypertension and type 2 diabetes in the Korean population (Kim *et al.,* 2001). Adelman etal.2001 followed seven African American adolescents who were identified with obesity-associated proteinuria. Those adolescents were grossly obese with average BMI 46 compared to our subjects whose BMI average was37. In addition, those children were mildly hypertensive and showed evidence of pathological renal changes.

So it is noticeable that in these studies which linked microalbuminuria to obesity the subjects may be obese but with other disorders that may cause renal injury egT2DM, hypertension or features of metabolic syndrome

It seems obesity uncomplicated by other risk factors eg. diabetes, hypertension and advancing age is not a cause of microalbumuria.

12.1 % of all our subjects showed evidence of microalbuminuria. A good number of cross- sectional and epidemiological studies have shown several disparities in the prevalence of microalbuminuria among different populations (Arghese *et al.,* 2001). These studies were mainly on patients with T2DM. These variations may reflect different stages of renal impairment induced by T2DM. However, Studies from Nigeria reported a high a high prevalence of microalbuminuria of about 33% among school children (Okpere *et al.,* 2012). Only 5% of the subjects were suffering from risks that known to cause microalbuminuria eg. Diabetes, hypertension and sickle cell anemia. This may suggest the possibility of ethnic variations as indicated by Jones *et al.* (2002) from their studies of microalbuminuria prevalence among Americans from different ethnic backgrounds.

A limitation of our study that a single spot urine sample was used for the measurement of albumin creatinine ratio which although widely used and accepted it may overestimate the prevalence of microalbuminuria

(Konta *et al.,* 2006)

We conclude that obesity by itself in otherwise healthy young adult males is not a cause of microalbuminuria. Detection of microalbuminuria in such subjects should call for further investigation for an underlying pathology.

ACKNOWLEDGEMENTS

We wish to acknowledge the help of Dr Humeda Sweket Ahmed for the help with the statistical analysis.

Conflict of Interest: No conflict of interest

REFERENCES

Adelman RD, RestainoIG, AlonUS, Blowey DL (2001). Proteinuria and focal segmental glomerulosclerosis in severely obese adolescents. J Pediatr. Apr;138(4):481-5,.

Agnani S, Vachharajani VT, Gupta R, Atray, NK, Vachharajani TJ (2005). Does treating obesity stabilize chronic kidney disease? BMC Nephrol. Jun 15;6:7,.

Arghese A, Deepa R, Rema M, Mohan V (2001). Prevalence of microalbuminuria in type 2 diabetes mellitus at a diabetes centre in southern india. Postgraduate Med. J. 77(908), 399,.

Bhurosy T, Jeewon R (2014). Overweight and Obesity Epidemic in Developing Countries: A Problem with Diet, Physical Activity, or Socioeconomic Status? *The Scientific World Journal.*;2014:964236. doi:10.1155/2014/964236

Brantsma AH, Atthobari J, Bakker SJ,Zeeuw D, Jong PE, Gansevoort RT (2007). What predicts progression and regression of urinary albumin excretion in the nondiabetic population? J Am SocNephrol, 18:637–645..

Bullo M, Garcia-Lorda P, Megias I, Salas-Salvado J (2003). Systemic inflammation, adipose tissue tumor necrosis factor, and leptin expression. Obes Res,11:525-531, ,

Chen B, Yang D, Chen Y, Xu W, Ye B, Ni Z (2010). The prevalence of microalbuminuria and its relationships with the components of metabolic syndrome in the general population of China. ClinChimActa. May 2;411(9-10):705-9.

Coresh J,Selvin E, Stevens LA (2007). Prevalence of chronic kidney disease in the United States. JAMA, 298:2018–2047.

Hasit D, Vasudha K (2012). A study of the levels of urinary microalbumin in non-diabetic normotensive obese individuals. *Advances in Biological Chemistry*, 2, 171-175.

Hoffmann IS, Jimenez E, Cubeddu LX (2001). Urinary albumin excretion in lean, overweight and obese glucose tolerant individuals: its relationship with dyslipidaemia, hyperinsulinaemia and blood pressure.J Hum Hypertens. Jun;15(6):407-12,

Jafar TH, Chaturvedi N, Hatcher J, Levey AS (2009). Use of albumin creatinine ratio and urine albumin concentration as a screening test for albuminuria in an Indo-Asian population. Nephrol Dial Transplant, 22:2194–2200,.

Jones CA, Francis, ME, Eberhardt, MS, et al. (2002). Microalbuminuria in the US population: Third National Health and Nutrition Examination Survey (NHANES III) Am J Kidney Dis. 39:445–459,.

Kaila B, Raman M (2008). Obesity: A review of pathogenesis and management strategies. Canadian Journal of Gastroenterology, 22(1), 61–68.

Kambham N, Markowitz GS, Valeri AM, Lin J, D'Agati VD (2001). Obesity-related glomerulopathy: an emerging epidemic. Kidney Int. 59 (4): 1498-509.

Kim YI, Kim CH, ChoiCS, ChungYE, LeeMS, LeeSI, ParkJY, HongSK, Lee KU (2001). Microalbuminuria is associated with the insulin resistance syndrome independent of hypertension and type 2 diabetes in the Korean population. Diabetes Res ClinPract 52: 145-152,

Konta T, Hao Z. Abiko H (2006). Prevalence and risk factor analysis of microalbuminuria in Japanese general population: theTakahata study. Kidney Int. ;70(4):751–756,.

Koroshi A (2007)Microalbuminuria, is it so important? Hippokratia. Jul-Sep; 11(3): 105–107.

Kovesdy CP, Furth SL, Zoccali C (2017). On behalf of the World Kidney Day Steering Committee. Obesity and Kidney Disease: Hidden Consequences of the Epidemic. Canadian Journal of Kidney Health and Disease.;4:2054358117698669. doi:10.1177/2054358117698669.

Lin WY, Pi-Sunyer FX, Liu CS, Li CI. Davidson LE, Li TC. et al. (2012). Central Obesity and Albuminuria: Both Cross-Sectional and Longitudinal Studies in Chinese. PLoS ONE 7(12): e47960. https://doi.org/10.1371/journal.pone.0047960

Nagwa MA, Elhussein AM, Azza M, Abdulhadi NH (2011). Alarming high prevalence of overweight/obesity among Sudanese children. European Journal of Clinical Nutrition, 65, 409–411,

Okpere AN, Anochie, IC, Eke FU (2012). Prevalence of Microalbuminuria among secondary school children Afr Health Sci. Jun; 12(2): 140–147,.

Poirier P, Giles TD, Bray G.A, Hong Y, Stern JS, Pi-Sunyer FX, Eckel RH (2006). Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss. ArteriosclerThrombVasc Biol. May;26(5):968-76,.

Preventing chronic diseases: A vital investment. World Global Report. Geneva: World Health Organization; 2005.

Rifkin DE, Katz R, Chonchoi M (2003). Albuminuria, impaired kidney function and cardiovascular outcomes or mortality in the elderly. Nephrol Dial Transplant, 25(12):1560–1567.

Seals DR, Jablonski, KL,Donato AJ (2011). Aging and vascular endothelial function in humans ClinSci (Lond).120(9): 357–375

Sonta T, Inoguchi T, Tsubouch, H, Sekiguchi N, Kobayashi K, Matsumoto S, Utsumi H, Nawata H (2004). Evidence for contribution of vascular NAD(P)H oxidase to increased oxidative stress in animal models of diabetes and obesity. Free RadicBiol Med 37:115-123,.

US Renal Data System, USRDS. (2005). Annual data report: atlas of end-stage renal disease in the United States. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases;.

Yesim TE, Ugurlu S, Caglar E, Balci H, Ucgul A. et al. (2007). Investigation of microalbuminuria in nondiabetic, normotensive obese women. Internal Medicine, 46, 1963-1965